QT50 could be the better marker of arrhythmogenic risk. Conclusions: A high incidence of QT prolongation was observed in psychiatric patients receiving psychotropic drugs and 24-hr Holter ECG was useful in evaluating bradycardia-dependent QT prolongation.

POSTER SESSION

1041 Autonomic and Central Nervous System Regulation of the Heart

Sunday, March 17, 2002, Noon-2:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1041-103 Induction of Cardiac Nerve Sprouting and Sympathetic Hyperinnervation by Subthreshold Electrical Stimulation of the Left Stellate Ganglion in Dogs

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Background: Subthreshold electrical stimulation in the brain can induce nerve sprouting and the kindling model of epilepsy. Whether or not subthreshold electrical stimulation can induce cardiac nerve sprouting is unclear.
Methods: Six dogs were used in the study. The chest was opened from the left 4th intercostals space. An active fixation pacemaker lead was screwed into the left stellate ganglion (LSG). The lead was connected to a Medtronic Itrel neurostimulator (N-=3) or a modified Guidant Discovery pacemaker (N=3) to give rapid stimulation at 20 Hz (0.45 ms pulse width) and 5 Hz (1.9 ms pulse width), respectively. We first determined the stimulation threshold (the lowest voltage output that produced an abrupt increase of heart rate of > 20% from the baseline). The pacemaker output was then programmed to 25% of the stimulation threshold for continuous subthreshold electrical stimulation for 41±9 days. The atrial and ventricular tissues were than harvested and stained for nerve markers tyrosine hydroxylase (TH), synaptophysin (SYN) and growth-associated protein 43 (GAP43) by immunocytochemical techniques. Tissues from 6 healthy dogs were used as controls.
Results: The densities (μm^2 per mm^2) of nerves that stained for TH, SYN and GAP43 were significantly (P<0.01) higher in dogs with LSG subthreshold electrical stimulation compared to matched controls (1657± 614, 6518± 6060, 4482± 1955 versus 635±339, 263±340, 359±296 for left ventricle; 2343±1415, 2186±791, 2579±1113 versus 502±268, 82±26, 434±269 for right ventricle; 12334±11128, 11270±7245, 15300±6473 versus 1082±1171, 631±605, 170±267 for left atrium and 16116±12553, 20040±10730,26388±11012 versus 2017±1349, 423±358, 650±1112 for right atrium, respectively). The nerve sprouting magnitude was significantly higher in the atrium compared to the ventricles (P<0.02). Furthermore, the heart weight of LSG group was 20±14% greater than of the control dogs (P<0.02).
Conclusions: LSG subthreshold electrical stimulation induces cardiac hypertrophy and sympathetic nerve sprouting. These effects are more prominent in the atrium than the ventricles.

1041-104 Catheter Stimulation of Cardiac Parasympathetic Nerves in Man: A Novel Technique

Karl Mischke, Patrick Schauerte, Christian Knackstedt, Markus Zarse, Anil Sinha, Thomas Schimpf, Christoph Stellbrink, Peter Hanrath, *Rheinisch-Westfälische Technische Hochschule, Aachen, Germany.*

Background: Cardiac parasympathic nerves run alongside the superior vena cava (SVC) and accumulate epicardially adjacent to the coronary sinus (CS) orifice. In animals transvascular catheter stimulation of these nerves results in a negative chronotropic and dromotropic effect without negative inotropy. The present study reports on the first experience with transvascular human parasympathetic nerve stimulation (PS) in the SVC and CS.
Methods: During electrophysiologic studies of 23 patients PS was performed in the SVC (n=13) or in the proximal CS (n=10). A deflectable multipolar electrode catheter was positioned in the SVC just above the atrial junction or into the proximal CS. PS in the SVC was performed with a frequency of 20 Hz and stimulation voltages of 10, 20 and 30V during sinus rhythm and during incremental right atrial pacing with a nerve stimulation voltage of 30V. During PS in the proximal CS, atrial myocardial tissue stimulation by high frequency nerve stimuli had to be avoided. We therefore applied trains of nerve stimuli (200 Hz, 50 ms train duration) within the atrial refractory period. The antegrade Wenckebach period was determined to assess a negative dromotropic effect.
Results: PS in the SVC caused a significant increase in heart rate depending on the stimulation voltage as well as a significant increase of the antegrade Wenckebach cycle length. PS in the CS led to a voltage-dependent increase of the antegrade Wenckebach cycle length until AV-block III occurred in 7 patients. The negative chronotropic and dromotropic effects started/ceased immediately after the onset/end of nerve stimulation and were abolished by atropine. Patients reported on moderate chest discomfort during nerve stimulation.
Conclusions: Transvascular electrical stimulation of human cardiac parasympathetic nerves can be achieved with conventional electrode catheters positioned in the SVC or CS. Using this novel stimulation technique, readily reversible negative chronotropic and dromotropic effects can be obtained. PS may be used for acute ventricular rate slowing during supraventricular tachycardias in patients with congestive heart failure or as a diagnostic tool during electrophysiological studies.

1041-105 First Evidence of Existence of a Localized Brainstem Center of Cardiovascular Control in Humans

Vladimir Shusterman, Irmute Usiene, Benhur Aysin, Maksim Glukhovskoy, Peter J. Jannetta, *University of Pittsburgh, Pittsburgh, Pennsylvania, Allegheny General Hospital, Pittsburgh, Pennsylvania.*

Functional relations between human brainstem and cardiovascular system are complex, and existence of a localized brainstem center of cardiovascular control (BC) in humans has been debated. Here, we present the first evidence of existence of a localized BC in humans using mechanical stimulations of the surface of the brainstem at multiple sites. **Methods.** The ventrolateral surface of the medulla oblongata was exposed in 8pts (age: 54±14y, 5male) undergoing neurosurgery, 3-7 equidistant sites were determined using metric paper, and at each site sequential mechanical stimulation of the brainstem surface was performed (frequency: 0.5-2Hz, duration:1min) using a 2-mm metallic ball. Spatial changes in cardiac repolarization were examined using 32-lead/192-site electrocardiographic body surface potential maps. Blood pressure was monitored using intra-arterial line. **Results.** During the stimulation between the caudal rootlets of the 10th nerve (Figure), the peak T-wave amplitude decreased (normalized difference:22%, range:6-50%, p=.025) and RR-intervals became shorter (from 923±190 to 794±111ms, p=.063) compared to the recordings obtained before the stimulation. QT-interval and the areas under the QRS and ST-segment did not change (p=.89,.78, and .40, respectively). **Conclusions.** Stimulation of a localized region of the ventrolateral surface of the brainstem between the caudal rootlets of the 10th nerve elicits pronounced effects on cardiac rhythm and repolarization.

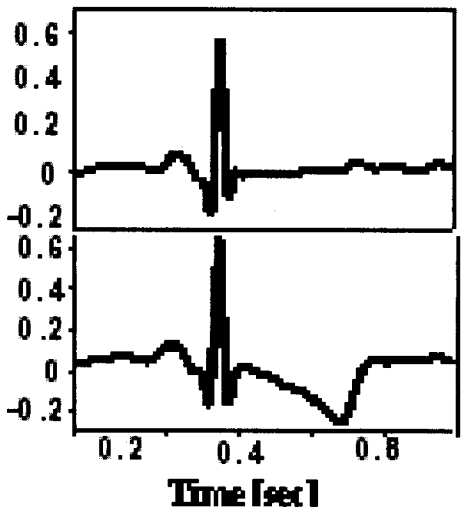

Figure. ECG in lead V4 before (top) and after the stimulation

1041-106 The Effect of Spinal Cord Stimulation on Autonomic Activity and Cardiac Nitric Oxide Overflow

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Background : Because spinal neurons influence intrinsic cardiac neurons, which in turn have their effects modulated by nitric oxide (NO) concentration, we measured the influence of spinal cord stimulation (SCS) on autonomic modulation of cardiac properties and coronary sinus (CS) NO.
Method : We measured the effect of SCS on spontaneous sinus cycle length (SCL) , atrioventricular (AV) nodal conduction time (AH interval during atrial pacing at CL 350-400msec.) and CS NO concentration in 5 groups of normal, open chest dogs anesthetized with alpha chloralose.
Result : SCS with intact autonomics resulted in increase in both BCL and AH (all values in msec, n=18; BCL from 541±2 to 607±2; AH from 77±6 to 84±6, p<0.01). SCS with stellate ganglion transection augmented the vagal-induced increase in BCL and AH (n=7; BCL from 683±3 to 707±2; AH from 103±7 to 112±7 , p<0.01) and blunted the sympathetic-induced shortening of BCL and AH (n=9; BCL from 331±1 to 396±1; AH from 52±5 to 61±4 msec, p<0.01) . Whereas SCS with vagal transection did not affect the sympathetic-induced shortening of BCL or AH (n=6; BCL from 344±4 to 345±4; AH from 70±7 to 70±7) nor the vagal-induced increase in BCL and AH (n=9; BCL from 696±3 to 699±2; AH from 102±1 to 103±1).
NO blood levels did not change significantly with SCS in any group. (n=40; 121.8 ±31.3 control and 117.2± 28.8 during SCS in nmol/min, p=0.58,) .
Conclusion; SCS augments vagal influences on the heart and can modulate (reduce) sympathetic influence on the heart by augmenting vagal activity and/or reducing sympathetic activity.